Presentation at the Aerospace Medical Association, Miami, Florida, 5/11/64.

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HYPOXEMIA INDUCED BY SUSTAINED FORWARD ACCELERATION IN PILOTS' BREATHING PURE OXYGEN IN A FIVE POUNDS PER SQUARE INCH ABSOLUTE ENVIRONMENT

NASA TMX 51649

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By W. C. Alexander*, R. J. Sever**, and F. G. Hoppin**

Manned spacecraft returning to earth following lunar expeditions will approach the earth's atmosphere at an approximate velocity of 37,000 feet per second. Successful return of man from space may depend largely on his tolerance to the forces of prolonged deceleration produced as the kinetic energy of the spacecraft is dissipated by aerodynamic drag upon entry. The magnitude and duration of accelerations characteristic of the entry phases of present and subsequent extended duration mission will require man to perform near the limit of his physical tolerance.

The debilitating effects of prolonged acceleration on the cardiovascular system have been minimized by orienting the crew transverse to the resultant vector of the decelerative force. However, tolerance to acceleration applied transverse to the crew is limited by inability of the cardiopulmonary system to maintain perfusion of the brain and body tissues with oxygen-rich blood. Respiratory involvement, consisting of alterations in lung volumes, the mechanics of breathing, and in the ventilation-perfusion relationship of the lungs under acceleration manifested primarily by a decreased ability of the lungs to oxygenate the mixed venous blood. The present investigation was undertaken to study the patterns and severity of the hypoxemia induced by such prolonged forward acceleration while breathing pure oxygen at a pressure of one-third atmosphere (5 psia). This investigation, while evaluating the

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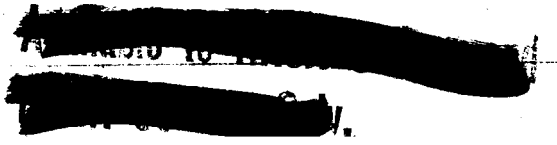

the tolerability of the space crew to simulated dynamic and environmental conditions of present manned earth entry missions, also brings to point additional relevant considerations concerning the mechanisms involved in hypoxemia induced by prolonged forward acceleration.

Review of Literature

Abnormal respiratory physiology resulting from sustained forward acceleration has been described previously by several investigators. Cherniack[1] and associates reported a significant increase in respiratory frequency, and an increase in inspiratory minute volume which was not associated with an increase in tidal volume. These changes were progressive with increasing magnitudes of acceleration. Zechman[11] reported a linear decrease in vital capacity with increasing levels of acceleration. Watson, et. al.[6], reported a proportional decrease in all lung volumes, with the exception of residual capacity which under acceleration increased relatively to the total lung volume. In support of these observations, Hershgold[2] demonstrated in lateral thoracic roentgenograms of humans undergoing exposures to prolonged forward acceleration, an increased radiolucency of the anterior lung fields, and an increased radiodensity of the posterior segments of the lungs. This investigator attributed these findings to an apparent pooling of the pulmonary blood in the dependent areas of the lung and a diminished vascularity of the anterior segments under acceleration.

The combined effects of these alterations in cardiopulmonary function on tolerance to forward acceleration are perhaps most evident in the demonstration of a decreased saturation of the arterial blood

under acceleration by several investigators. Wood and associates[10] demonstrated that the resultant level of oxygen saturation was dependent upon the magnitude of the acceleration, the duration of the exposure, and the oxygen content of the air breathed. Exposures in excess of 250 seconds at 5g with the pilot breathing ambient air resulted in arterial saturation levels of 81 percent, reaching this level within 100 seconds following the onset of acceleration. Recovery to prerun saturation values following termination of the exposure was prolonged. Nolan, et. al.[3], reported an average arterial oxygen saturation of approximately 88 percent following exposures of 190 seconds to 5.4 transverse g under similar conditions. Reed and co-workers[4] reported a resultant saturation of 72 percent following exposure to 7g. These investigators observed a constant rate of fall of oxygen saturation, independent of the magnitude of maximum g, which stabilized within 80 seconds of the onset of acceleration.

These investigators have, in general, postulated that the arterial oxygen desaturation observed under prolonged forward acceleration is the result of large venoarterial shunting in the lungs as the pulmonary blood flows past poorly ventilated or atelectatic alveoli and possibly pulmonary edema.

Further evidence of a defect in the ventilation-perfusion relationship in the lung during forward acceleration has been suggested by Wood and Nolan by demonstrating a less pronounced decrease in arterial oxygen saturation with the pilot breathing pureoxygen at ambient pressure rather than air. Wood and associates[10] noted that with the pilot breathing 99.6 percent oxygen, the fall in oxygen saturation during exposure to 5 transverse g

was negligible during the initial 30 seconds of the run and Nolan, et. al.[3], were unable to show a change in prerun saturation levels under acceleration of 3.7g sustained for 190 seconds. However, Steiner and Mueller[5] reported a resultant saturation of 86 percent following a 108 second exposure to 8 transverse g while breathing 100 percent oxygen.

Although many postulates have been advanced for the decrease in arterial oxygen saturation during prolonged forward acceleration, the relative value of each of these possible mechanisms has not been demonstrated.

Materials and Methods

The present investigation was conducted at the Aviation Medical Acceleration Laboratory (AMAL), Johnsville, Pennsylvania, jointly by Crew Systems Division of the NASA Manned Spacecraft Center and the Aerospace Medical Division of AMAL to measure the extent of hypoxemia induced by forward acceleration under conditions that simulate the dynamic and environmental aspects of manned earth entry missions. Thirty-five highly motivated, professional, military pilots, without previous centrifuge experience participated as experimental subjects in the investigation. Arterial oxygen saturation was measured directly by manometric analysis (method of Van Slyke-Neill) of samples withdrawn under sustained levels of forward acceleration and compared to the output of an earpiece oximeter. Ten pilot subjects participated in a correlation analysis whereby the output of the earpiece oximeter was calibrated to reflect the values of arterial oxygen saturation as determined by manometric analysis of arterial blood collected under

acceleration. The principal phase of the investigation consisted of the measurement of arterial oxygen saturation and relevant cardiopulmonary events under sustained forward acceleration. Twenty-five pilot subjects participated in this phase of the investigation.

a. Calibration of Earpiece Oximeter

Ten pilot subjects selected at random from the pilot population were given a thorough prerun physical examination and prepared in the following manner. The pilot's left forearm was surgically prepared for insertion of an arterial cannula into the brachial artery at the level of antecubital fossa. Following local infiltration with 1 percent Xylocaine of the superficial and deep tissues of the fossa, percutaneous entry into the vessel was made medial and approximately 1-2 cm above the lacertus fibrosus of the biceps muscle with a 17 ga. Rochester plastic needle.* The steel shaft of the needle assembly was withdrawn leaving the plastic sheath indwelling approximately 5 cm in the lumen of the artery. The Luer hub of the cannula was mated in series through a sterile three-way stopcock to an oiled ten cubic centimeter syringe, spring loaded to insure rapid and complete filling and containing heparin. The syringe assembly was secured to the forearm and hand to prevent movement under acceleration.

A snugly fitting, soft helmet liner was secured to the subject's head. An earpiece oximeter** was attached to the pilot's left ear and taped securely to the liner. Prior to attachment of the oximeter, the pinna of the ear was vigorously rubbed with an acetone sponge to

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* Model 503, Rochester Plastic Company, Rochester, Minnesota

** Model XE60-A, Waters Corporation, Rochester, Minnesota

to induce dilation of the superficial vasculature. The methods, applications, and characteristics of the oximeter assembly are described elsewhere.[8,9] The output of the oximeter was amplified logarithmically*** by the method first described by Wiederhielm[7]. An aviator's helmet**** was placed over the subject's ear piece assembly and secured to the head.

The pilot walked to the centrifuge platform and entered the gondola. He was restrained in a contoured fiberglas and metal couch in the supine-sitting position in the configuration described in figure 1. The pilot's left arm, bearing the blood sample collecting assembly, was restrained fully supinated at an angle of 20 degrees away from the midline at the level of the heart. Each pilot was instructed to open the stopcock to the syringe on command from the test conductor and was not required to perform other tasks during the centrifuge run. The investigation was conducted with the pilot breathing room air.

Pilot subjects with indwelling arterial cannulae were taken to peak acceleration levels varying individually from 2 through 10 g with onset rates of 1.5 g/sec. and held until the oxygen saturation had reached a stable value. After approximately 10-15 seconds of negligible change in the oxygen saturation as evidenced by analog recording of the output of the oximeter, the pilot was instructed to open the stopcock to the syringe and thereby collect the blood sample. At completion of filling of the syringe, monitored by closed-circuit television, the centrifuge

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*** Model OSA-2B, Ensco Corporation, Salt Lake City, Utah

**** Model APH-5, U. S. Navy Standard Issue

was brought to a controlled stop and the sample immediately collected from the support assembly. The syringe was agitated to insure thorough mixing with the Heparin and was sealed with mercury and placed into ice. Samples collected were immediately analyzed manometrically for oxygen content, oxygen capacity, carbon dioxide and pH.

Simultaneous analog recordings of the output of the earpiece oximeter were made during the arterial sampling procedure under acceleration. Using the results of manometry from 30 arterial samples collected under sustained forward acceleration as a standard, a calibration plot was established from which all subsequent measurements of arterial oxygen saturation were based. Previous investigations of Nolan, et. al.[3] have described the correlation between the earpiece oximeter (indirect method) and the cuvette oximeter (direct method) for measurement of arterial oxygen saturation under sustained forward acceleration. Results of their study demonstrate an average value determined by the earpiece oximeter of 3 saturation units below that determined by the cuvette oximeter when both instruments were operated on the double-scale principle under acceleration. Scaling the output of the earpiece oximeter to reflect the values of arterial oxygen saturation determined manometrically from samples collected under acceleration should eliminate this apparent disagreement in the two methods under dynamic conditions reported by Nolan.

b. Principal Investigation

Twenty-five pilots participated in the principal phase of the investigation. Following a prerun physical examination, electrocardiographic

leads were applied to the body in a modified lead II configuration (axillary) to record cardiac events. Respiratory rate was measured by impedance pneumography utilizing the axillary lead configuration. The earpiece oximeter was attached to the pilot's left ear and the subject restrained in the couch of the centrifuge gondola as previously described.

Two environmental conditions were employed in the investigation. Pilots breathed air at ambient pressure and 100 percent oxygen with the gondola evacuated to a simulated altitude of 27,000 feet (5 psia). All pilots were fitted with an aviator's face mask***** and breathed through a demand regulator from gas bottles located on the arm of the centrifuge. Pilot subjects participating in the investigations while breathing 100 percent oxygen at a pressure of 5 psia were preoxygenated for a period of 1 hour prior to dynamic testing.

Tidal volume and respiratory rate were measured by a gas flowmeter****** placed in the air/oxygen line between the demand regulator and the pilot's face mask. Figure 2 schematically represents the mechanics of gas supply to the pilot. The output signal of the flowmeter was fed through an integrating electronic circuit and minute volume was recorded.

Pilot subjects were taken to peak acceleration levels along double pulse, square-wave profiles with onset rates of 1.5g/sec. Combinations of initial, intermediate, and secondary levels of acceleration were employed to simulate a velocity dissipation of 37,000 ft/sec in these profiles. Representative profiles are portrayed in

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***** Model A-14-A, U. S. Navy Standard Issue

****** Gulton Industries, Metuchen, New Jersey

figures 3-6. Pilots were allowed to rest following each profile for approximately 15 minutes prior to the next run or until the oxygen saturation recovered to prerun levels. A total of four profiles was experienced by each pilot under dynamic conditions within a 3-hour period. The exposures were so planned as to alternate high level profiles with those of low levels in an attempt to minimize fatiguing effects of runs earlier in the sequence. Prior to each dynamic run, control physiological data were collected and recorded for each pilot.

Following the completion of the sequence of static and dynamic centrifuge runs, each pilot underwent a postrun physical examination and participated in a comprehensive debriefing session at which time subjective impressions of each pilot under the conditions of the investigation were recorded.

Discussion of Results

Arterial oxygen saturation was measured in twenty-five pilot subjects during sustained forward acceleration conditions that simulate the dynamic and environmental aspects of present manned earth entry missions. Representative double pulse acceleration profiles were employed to combine both operational similarity and controlled investigative procedures. In addition, two profiles typical of actual mission conditions were employed to relate these findings to operational situations.

Figures 3-6 compare the resultant patterns of oxygen saturation as a function of the magnitude and duration of a double pulse, square-wave

exposure to forward acceleration. These figures demonstrate a progressive decrease in arterial oxygen saturation with increasing levels of acceleration from 4 to 10 transverse g with pilots breathing pure oxygen at a simulated altitude of 27,000 feet (5 psia). By independently varying the magnitude of the initial and secondary pulses between 4 to 10g, and the magnitude of the intermediate level between 2 and 5g, various patterns of arterial desaturation and recovery are demonstrated.

The fall in arterial oxygen saturation is evident approximately 30 seconds following the onset of the initial acceleration pulse. The rate of fall in saturation is dependent upon the magnitude of the maximum acceleration reached. Stable levels of saturation are attained for the 4 and 6g exposures, however, the saturation is still falling at the end of the initial 8 and 10g pulses. Maximal mean desaturation varies directly as a function of the magnitude of the acceleration pulse. Recovery to resting levels is not reached in the 90-second intermediate period, however the extent of recovery varies as a function of the interim level of acceleration. The onset of the secondary acceleration pulse in each instance produces a fall in saturation levels from their interim values to levels dependent on the magnitude of the second pulse. Recovery from the second plateau of the profile is complete in all cases approximately 120 seconds following the end of the run and relatively independent of the magnitude of the acceleration. However, within the initial 30 seconds following the exposure, rate of recovery apparently varies as a function of the magnitude of the maximum level of acceleration.

Two operational acceleration profiles representative of a typical mission were employed in this phase of the investigation to measure the resultant oxygen saturation during exposures to high magnitude, short duration accelerative force, and to correlate the results recorded from double pulse, square-wave exposures to those recorded under simulated operational conditions. Figure 11 demonstrates the decrease in saturation resulting from a single haversine exposure of 15 transverse g. The rate of fall in saturation following onset of acceleration is comparable to those described for double pulse square-wave exposure; however, the rate of desaturation was maximal at the time of completion of the run, and the maximal mean desaturation predictable for this magnitude of acceleration was not reached. It is of interest to note that the fall in saturation is coincident with the downslope or termination of the acceleration profile. The recovery from the desaturation is comparable to those observed under square-wave exposures and is complete within 120 seconds of the end of the exposure. In Figure 12, we see perhaps the best correlation between the double pulse, square-wave and the simulated operational profiles. Following an initial, short duration exposure to 12 transverse g, the fall in oxygen saturation is apparently stabilized by the onset of the secondary plateau. The value of the maximal mean desaturation is comparable with those recorded from square-wave exposures of the same magnitude and duration. Recovery from maximum desaturation is consistent with the observations on square-wave exposures.

Pilot subjects were exposed to double pulse, square-wave and simulated operational profiles while breathing air at ambient pressure (approximately

14.7 psia) for comparison with the patterns recorded in the 5 psia oxygen environment. The fall-in oxygen saturation is seen 10 to 15 seconds earlier in this environment as compared to the pure oxygen environment (Figures 3-6). However, the rate of fall in oxygen saturation is again shown to vary as a function of increasing levels of acceleration. The discrepancy in the rate of desaturation at 8g as compared to 10g is considered an artifact and does not represent a significant finding. The patterns of desaturation observed in this environment are comparable to those demonstrated in the pure oxygen environment at reduced pressure. The level of maximal mean desaturation is dependent on the magnitude of acceleration, however, there is a decrease in the resultant levels of saturation following both the initial and the secondary acceleration levels. Recovery from the desaturation resulting from the initial level of acceleration is apparently enhanced in the air environment as evidenced by figures 3 through 6. It is also of interest to note in these figures that the fall in oxygen saturation from the interim level is earlier than that observed in the oxygen environment. The rate of recovery from maximal mean desaturation levels following the second acceleration plateau when the pilot is breathing air is more than two-fold than when he is breathing 5 psia oxygen. The time of recovery from maximal desaturation while he is breathing air is less than in the oxygen environment and was not shown to vary as a function of the level of acceleration.

The desaturation pattern while he is breathing air during exposures to the simulated operational profiles closely resembles that recorded in the

oxygen environment. However, as shown in figures 11 and 12, the fall in oxygen saturation while he is breathing air is evident approximately 15 seconds prior to the fall while breathing oxygen. The levels of maximal mean saturation in both instances are lower than that recorded in the oxygen runs. The onset of recovery, as shown in figure 12, was not as sharply defined as in the oxygen study. The recovery pattern from the initial acceleration level is apparently coincident with the stabilization level of the secondary plateau.

Despite the lower levels of maximal mean desaturation recorded in the exposure to forward acceleration when the pilot is breathing air, subjective tolerance was in most instances better than exposure to similar dynamic conditions when he was breathing oxygen.

In order to observe more closely the hypoxemia patterns under forward acceleration, pilot subjects were exposed to single pulse, square-wave profiles while they were breathing air and while breathing oxygen at a pressure of one-third atmosphere. The magnitude and duration of 8 and 10g single pulse profiles represented a velocity change of 37,000 ft/sec. The 4 and 6g exposures were terminated when stable levels of saturation were reached, and did not represent the total velocity decrement.

The patterns of hypoxia as a result of these exposures are represented in figures 13 and 14. The patterns of desaturation following onset of acceleration are similar to those observed during the double pulse exposures. The rate of fall in saturation varies as a function of increasing acceleration with the more precipitous drops coming with the higher g exposures (figure 7). At 4 and 6g, stable saturation levels are reached within 120 seconds of the onset of acceleration, but

at 8 and 10g, the saturation was still falling when the runs were terminated prematurely because of subjective discomfort. Following 120 second exposures to 8 and 10g, the maximum levels of desaturation reached were 84 and 82 percent, respectively, while the pilot was breathing oxygen at reduced pressure, and approximately 2 to 4 percent while he was breathing air. In the single pulse runs, the environment of the pilot made the difference. Maximum mean desaturation levels under the four levels of acceleration reported are lower by 2 to 4 percent while he was breathing air (figure 8). The patterns of desaturation have similar onset and progression.

The onset of resaturation is immediate following the end of the exposure. There is a sharp initial rise in saturation corresponding to deep breathing by the pilot following the end of the run. The rate of this rise is highest following the most severe desaturations. The resaturation pattern thereafter is gradual and prolonged, however under all conditions, 95 percent saturation is regained within 120 seconds following the end of the run (figure 9). Perhaps the most striking observation contrasting the progression of resaturation while the pilot was breathing air and while breathing oxygen is shown in figure 10. The rate of recovery following the initial 30 seconds of t the exposure is less acute while the pilot is breathing oxygen rather than air, which does not appear to be caused by a significantly lower saturation going into the recovery phase.

Minute volume and respiratory rate increase with increasing levels of acceleration with apparently insignificant variations between the air or oxygen breathing exposures. Quantitative analysis of these data was not considered contributory to this investigation, however, it is

of significance to note that the patterns of desaturation were observed in the presence of an increased inspiratory minute volume.

As a part of the results, a brief description is included of two unusual difficulties encountered during the investigation. Neither of these difficulties occurred in more than a few pilots but both were significant enough to be of interest.

Three pilots had to be removed from the centrifuge because of severe chest pain which was aggravated by deep breathing. This pain was not related to the substernal chest pain experienced by some subjects normally under forward acceleration. It occurred on these pilot's first exposure to acceleration and remained present following the centrifuge run. One of the pilots was exposed to acceleration 4 days later and did not experience the same difficulty. Attempts at taking a deep breath also induced coughing. Following exposure to acceleration, the arterial oxygen saturation remained low for periods up to 20 minutes despite vigorous coughing. Normally, immediately following exposure to acceleration, a deep breath or cough caused a very sharp, dramatic increase in arterial oxygen saturation. This did not occur in these pilots. Rather, the arterial saturation increased only very slowly back toward normal with no sharp rises in spite of deep breathing or coughing. One of these pilots had mild hemoptysis on the day following his run. It was felt that these pilots had developed atelectasis. X-rays, unfortunately, were not available immediately. Films taken 30 minutes to 1 hour following the run and after vigorous coughing failed to show atelectasis.

Another phenomenon observed during the resaturation following the exposure to acceleration in some pilots was a rather abrupt, spontaneous decrease in arterial oxygen saturation of 5 to 10 percent. This occurred with the pilots lying quietly on their backs in the centrifuge couch. The desaturation was easily reversed if the pilot took a deep breath or coughed. This would occur repeatedly in a susceptible pilot producing a wavy arterial saturation curve. The tendency for this to occur decreased with time and did not recur after 10 to 15 minutes rest. If the pilot then made another run, the same thing would happen in the recovery period. The only symptom during these periods of desaturation was an occasional report of a heavy feeling in the chest.

Another observation of interest is that oxygen saturation following acceleration can be prolonged voluntarily by the pilots not taking deep breaths or coughing. On several runs where the experiment was attempted, there was little resaturation until the pilot took a deep breath.

Concluding Remarks

The patterns of arterial oxygen desaturation induced by sustained forward acceleration observed when the pilots were breathing oxygen at 5 psia are similar to those recorded when they were in an environment of air at ambient pressure. The milder desaturations seen in pilots under forward acceleration when they were breathing oxygen are consistent with the higher oxygen tension inspired under these conditions. In the present investigation there was little subjective difference reported by the pilots between the two environments. Although the recovery pattarns were prolonged in pilots breathing oxygen, subjective reports of postrun discomfort and dyspnea were equally frequent relative to both conditions.

Acceleration profiles representative of simulated operational conditions were equally well tolerated in both environments. The single pulse, square-wave profiles were subjectively more difficult than the double pulse exposures, although both represent equal velocity change. This preference for the double pulse exposures is most likely attributable to the interim period of partial recovery in oxygen saturation at levels of acceleration less than 4g. It is doubtful that interim levels in excess of 4g will augment tolerance appreciably.

The findings of this investigation were consistent with the various postulated mechanisms of desaturation under acceleration, namely, atelectasis, ventilation-perfusion imbalance, and pulmonary edema. The clinical picture in the individual pilots mentioned, the dependence of resaturation on deep breathing, and the modification of the patterns of hypoxemia by breathing a fully reabsorbable gas appear most consistent with atelectasis.

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By W. C. Alexander, R. J. Sever, and F. G. Hoppin

ABSTRACT

The study of arterial oxygen saturation during sustained accelerations similar to those anticipated in the Apollo and subsequent earth entry missions has been conducted in a joint Manned Spacecraft Center-Aviation Medical Acceleration Laboratory program at the Johnsville centrifuge facility. Cabin environments of one atmosphere and one-third atmospheres, with air and 100-percent oxygen, respectively, breathed on demand by the pilot were employed in the study. Data collected for thirty-five professional military pilots demonstrate a resultant diminishing arterial oxygen saturation as a function of magnitude and duration of acceleration and the environment of the pilot.

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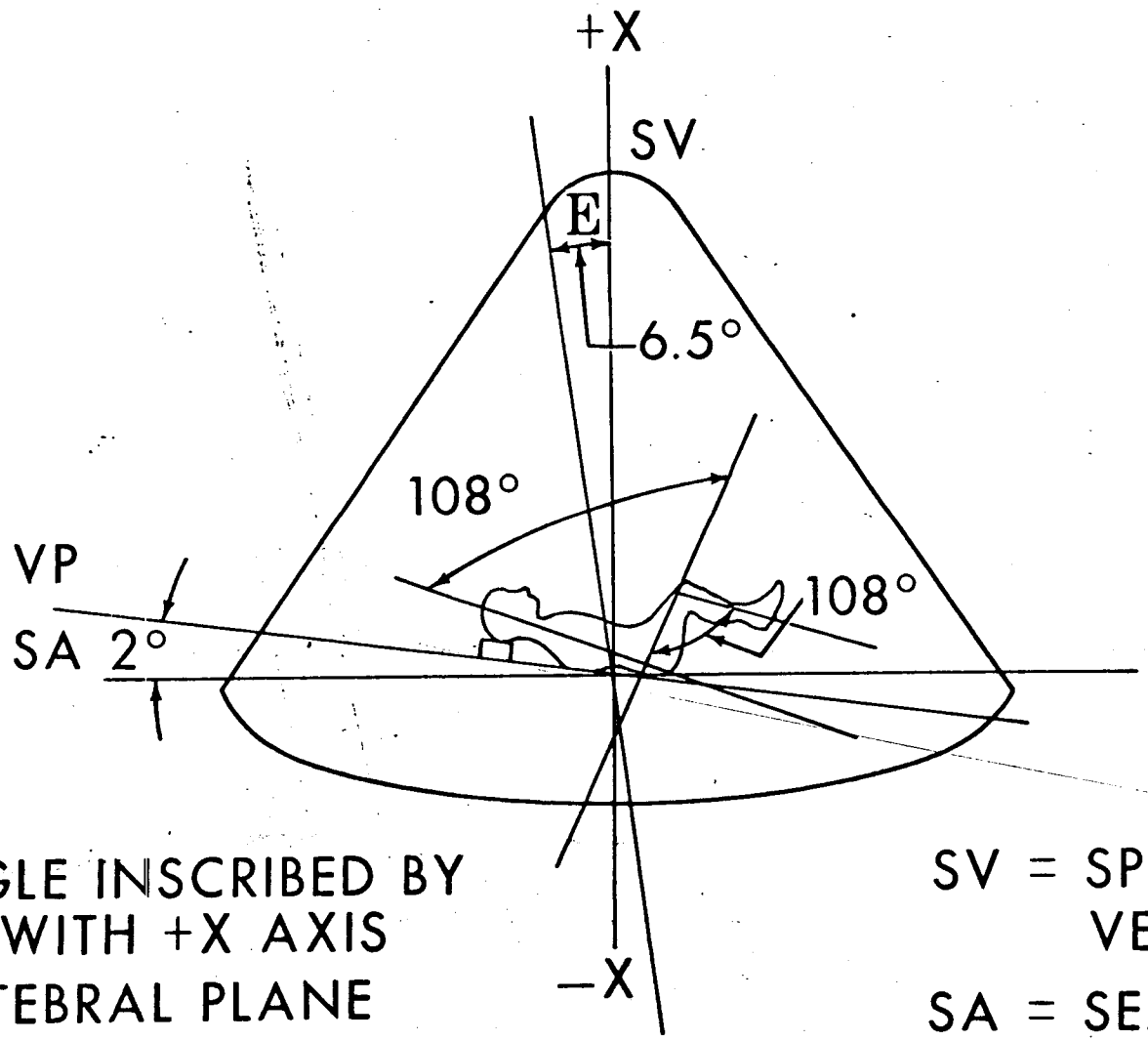
PILOT ORIENTATION



E = ANGLE INSCRIBED BY RFV WITH +X AXIS

VP = VERTEBRAL PLANE

RFV = RESULTANT FORCE VECTOR

SV = SPACECRAFT VERTICAL

SA = SEAT ANGLE

FIGURE 1

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MECHANICS OF GAS SUPPLY TO PILOT

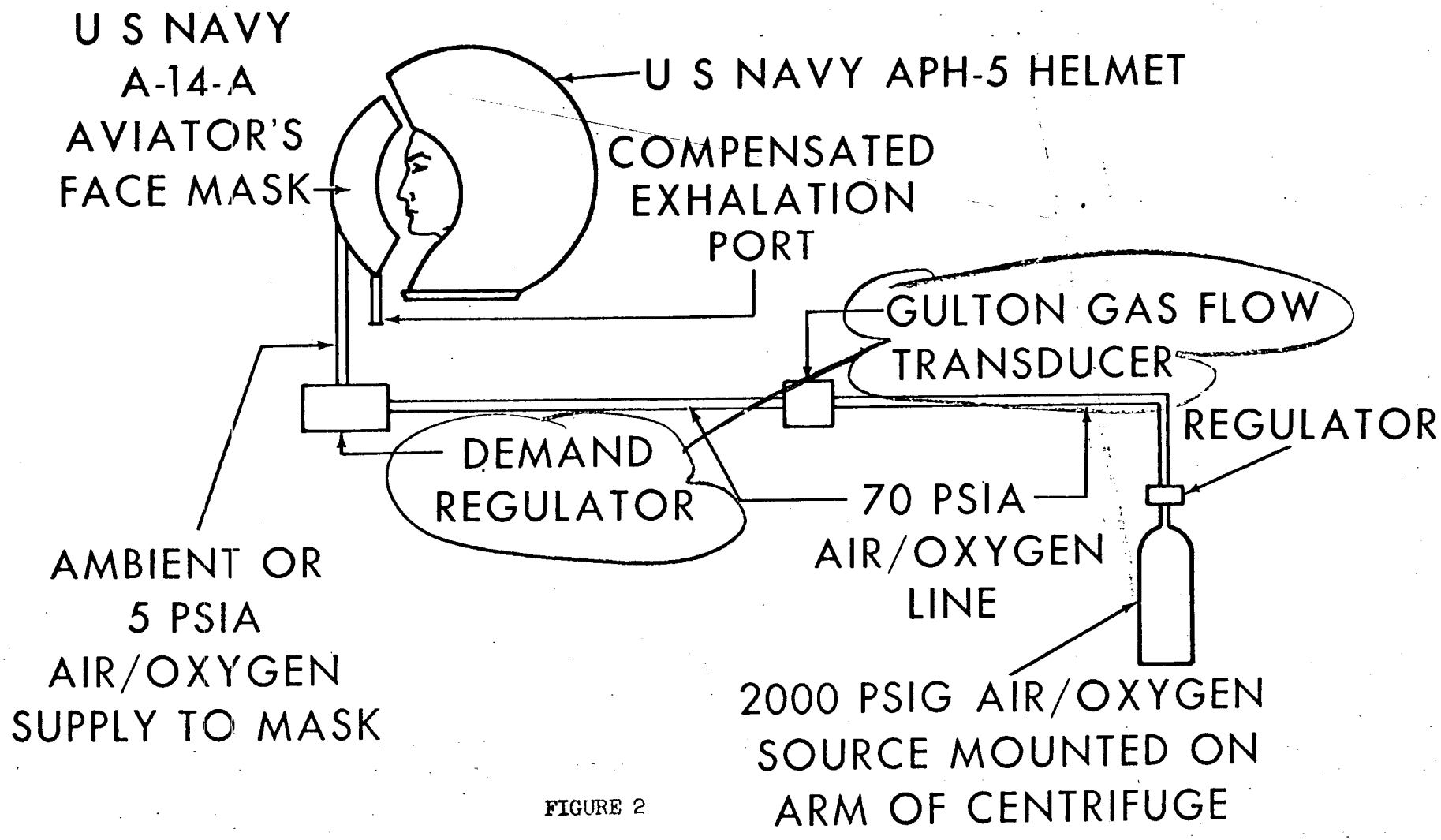

FIGURE 2

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ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION

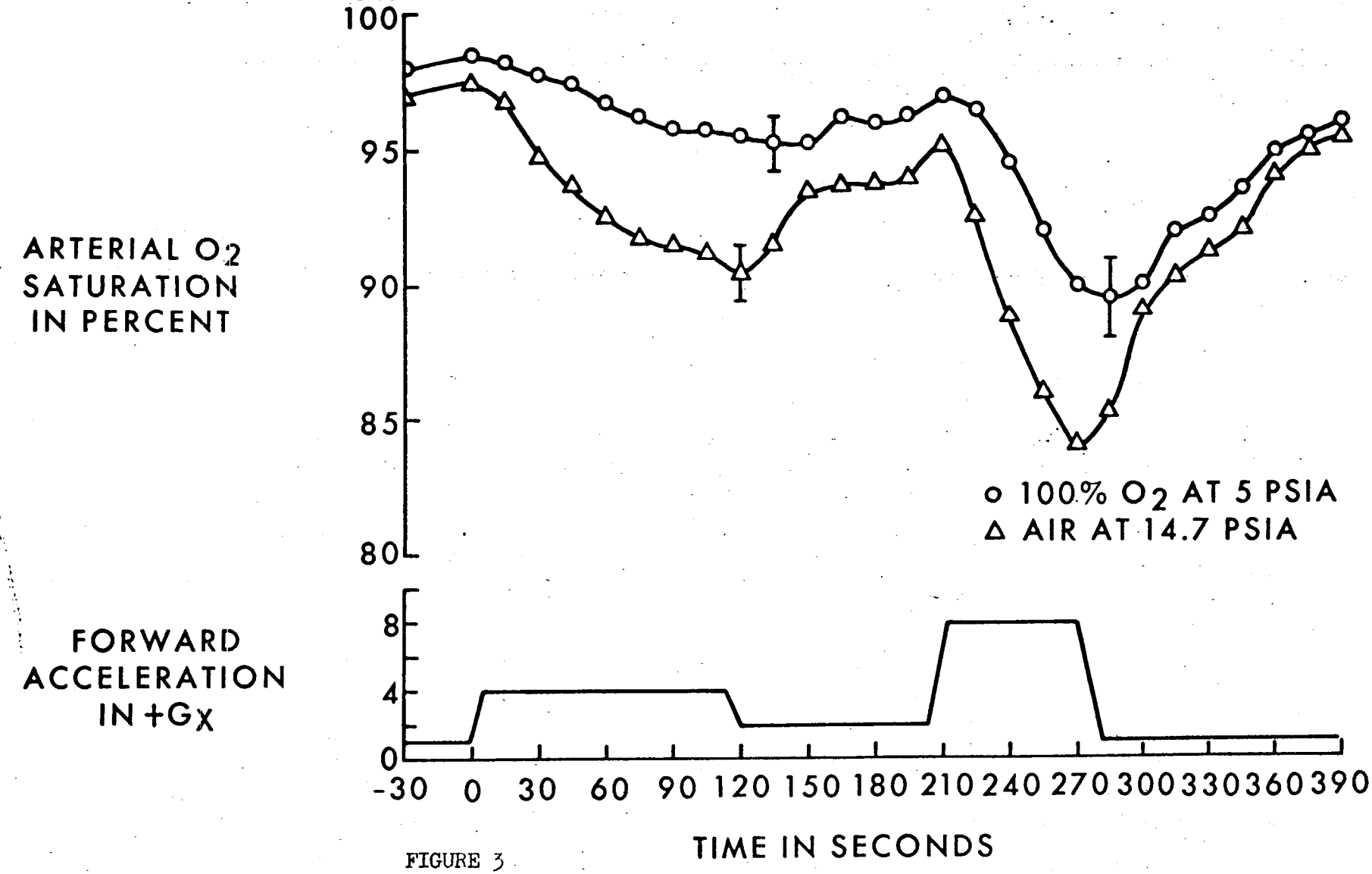

FIGURE 3

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ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION

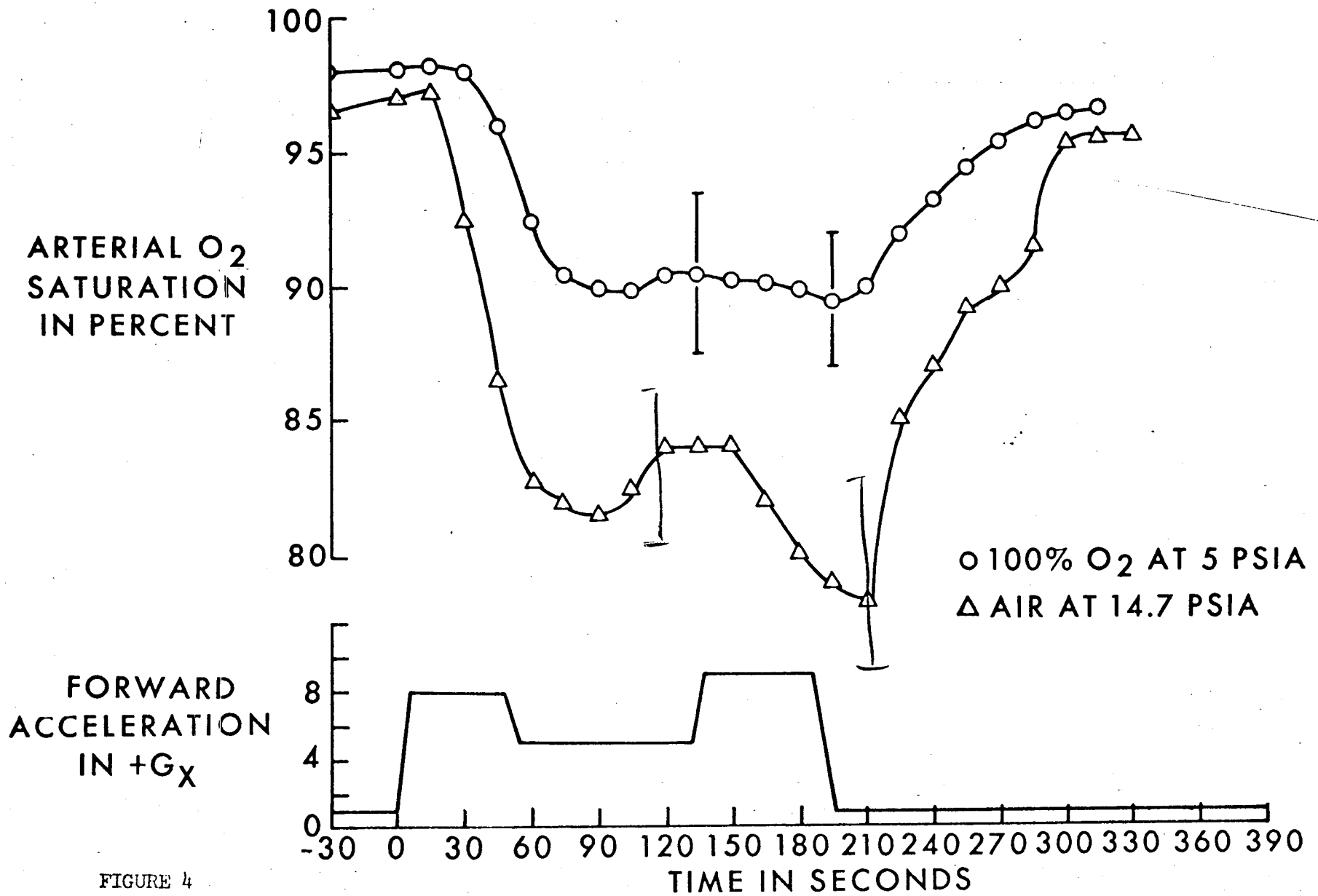

FIGURE 4

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ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION

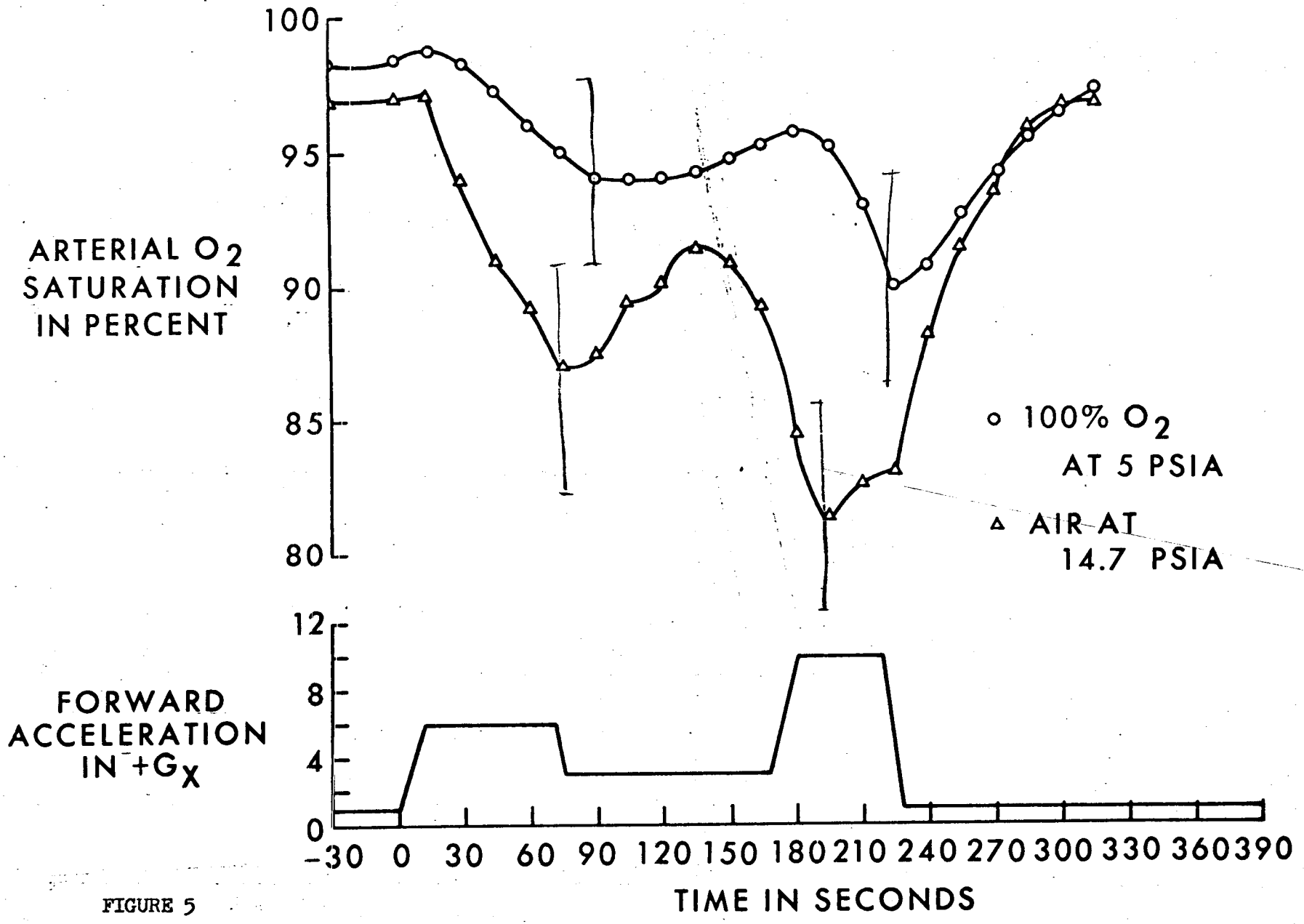

FIGURE 5

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ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION

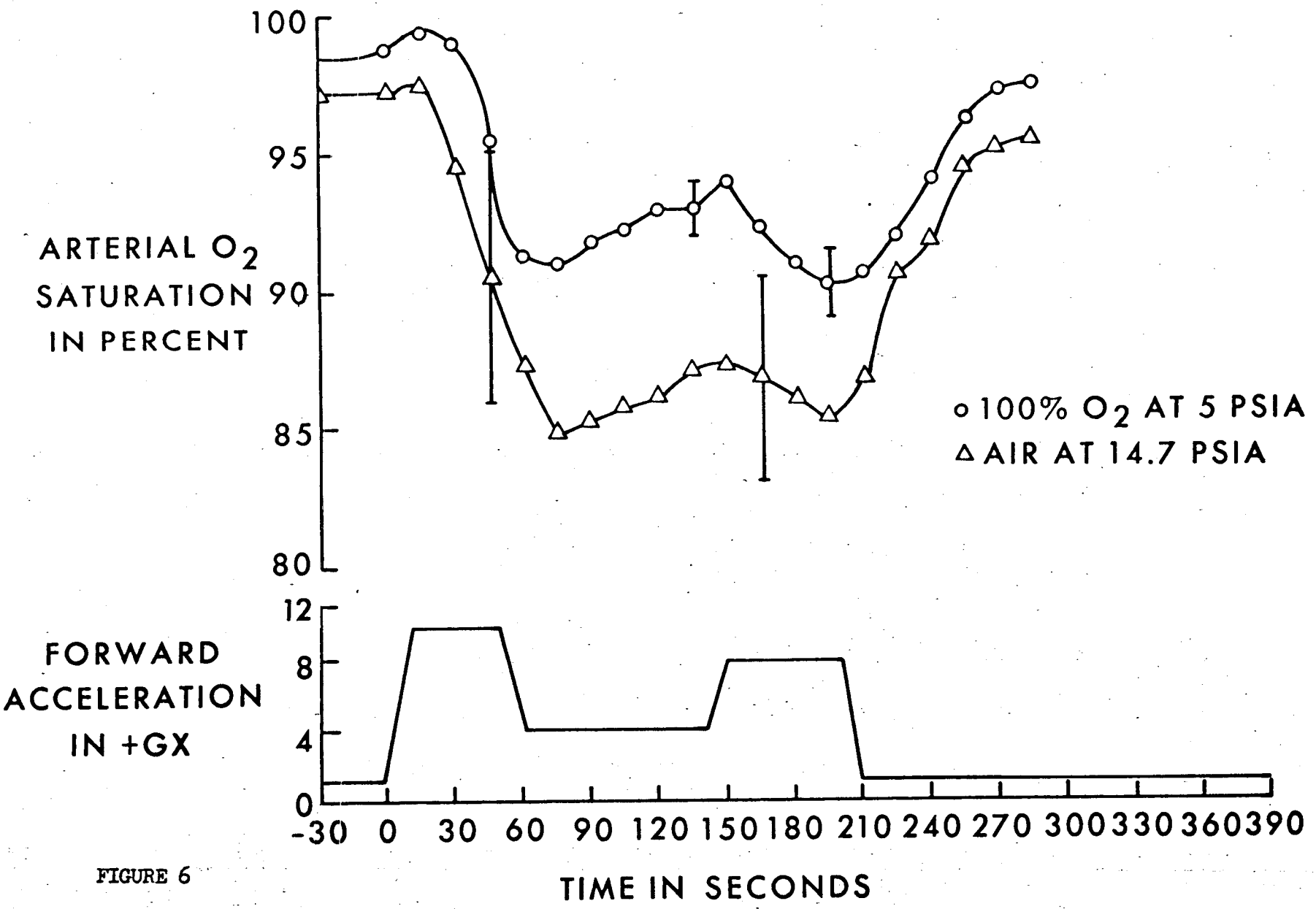

FIGURE 6

NASA-S-64-2605

PERCENT DESATURATION DURING TIME INTERVAL T+30 TO T+60 SECONDS

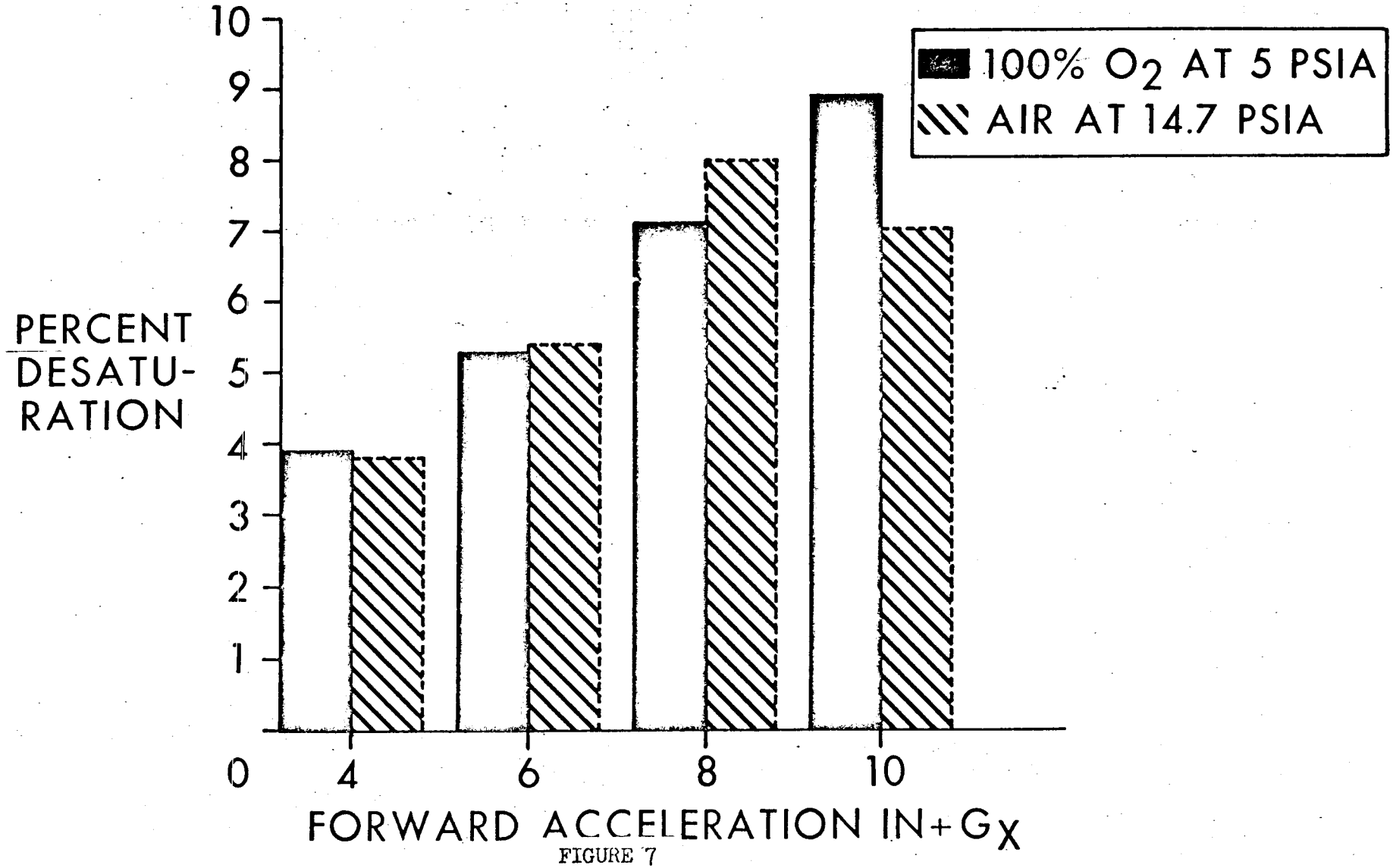

FIGURE 7

NASA-S-64-2606

MAXIMAL MEAN DESATURATION AT 120 SECONDS AFTER ONSET

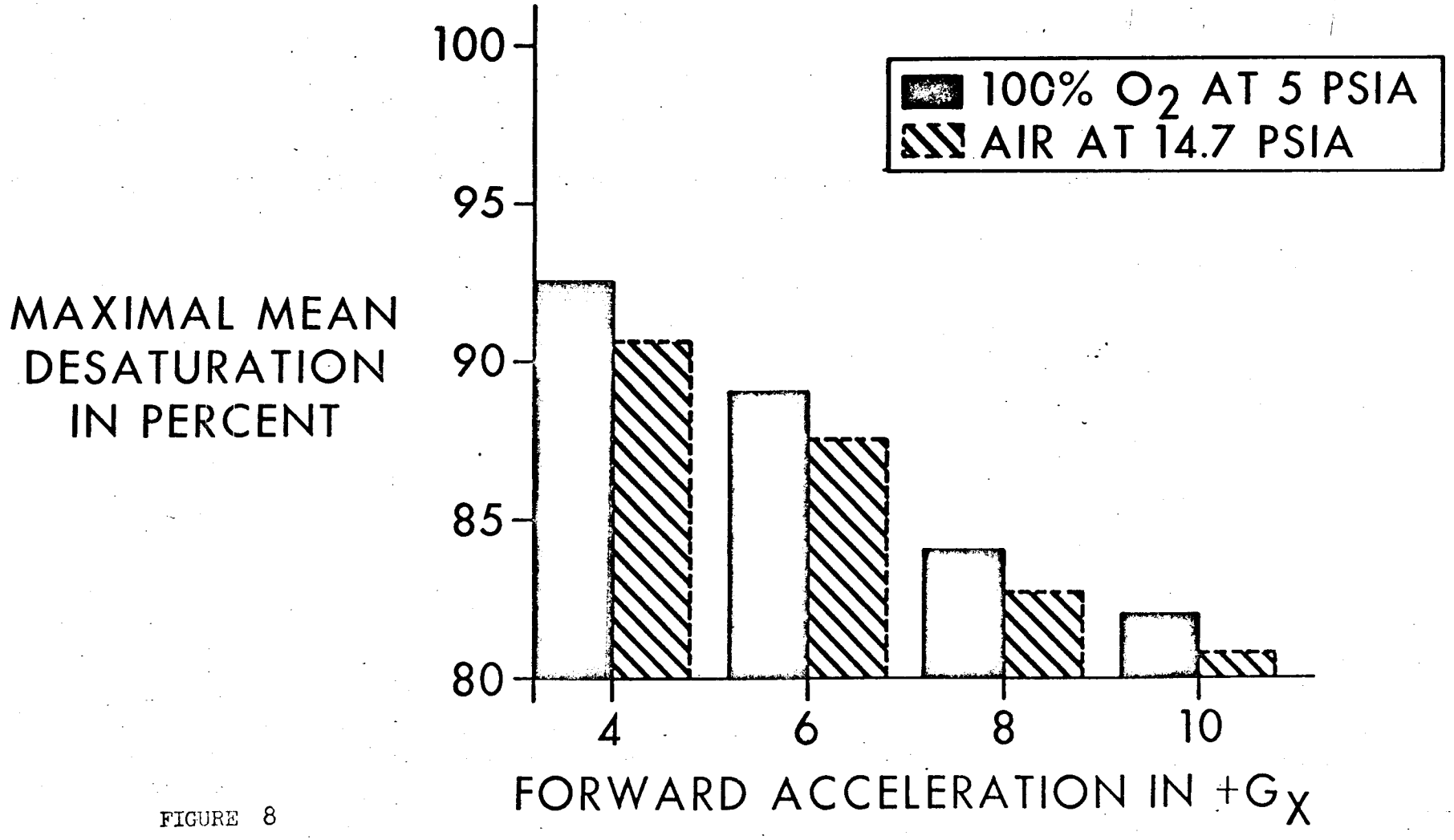

FIGURE 8

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TIME OF RECOVERY TO REGAIN 95% SATURATION

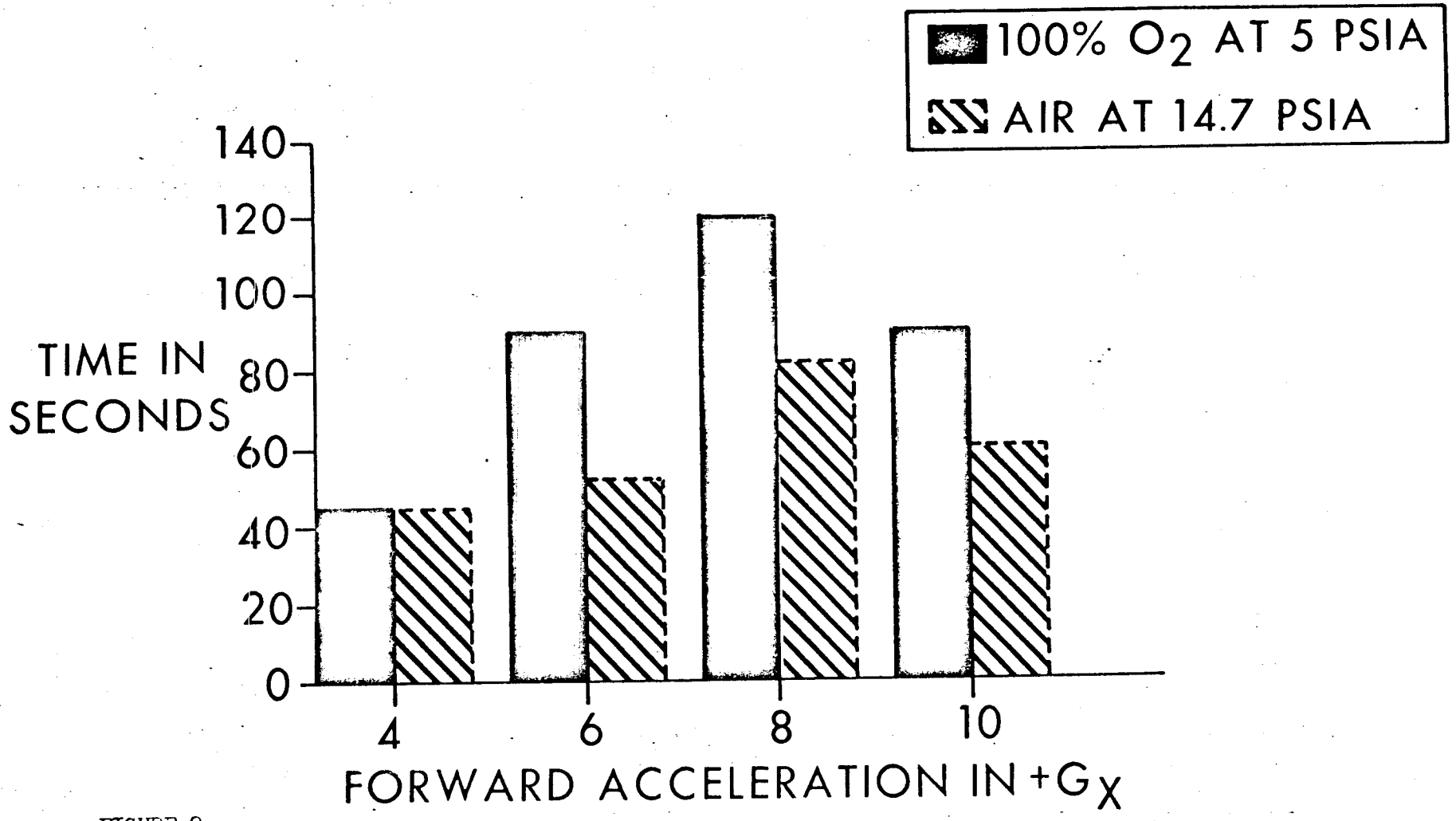


FIGURE 9

NASA-S-64-2608

PERCENT RESATURATION DURING INITIAL 30 SECONDS OF RECOVERY

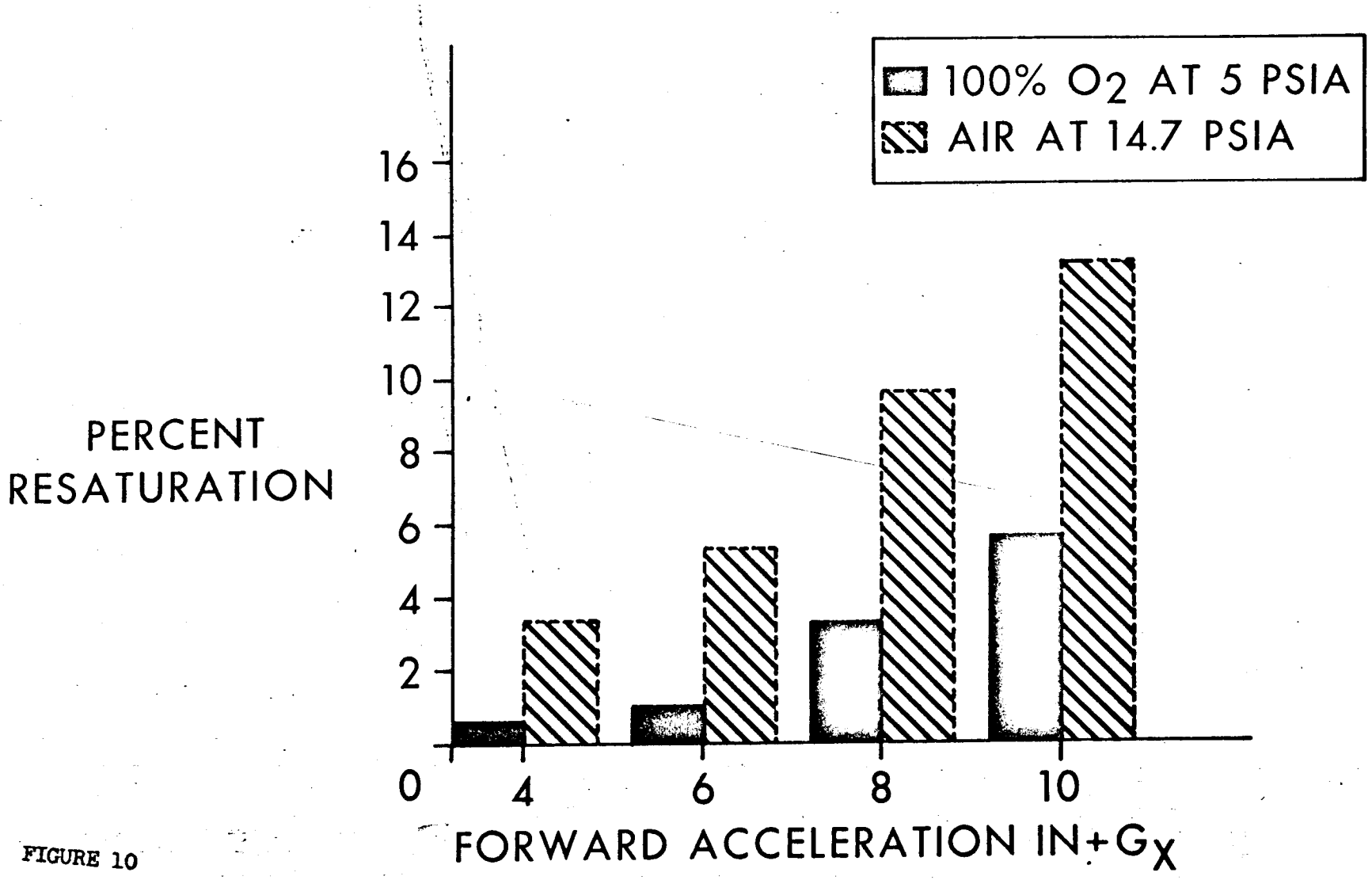
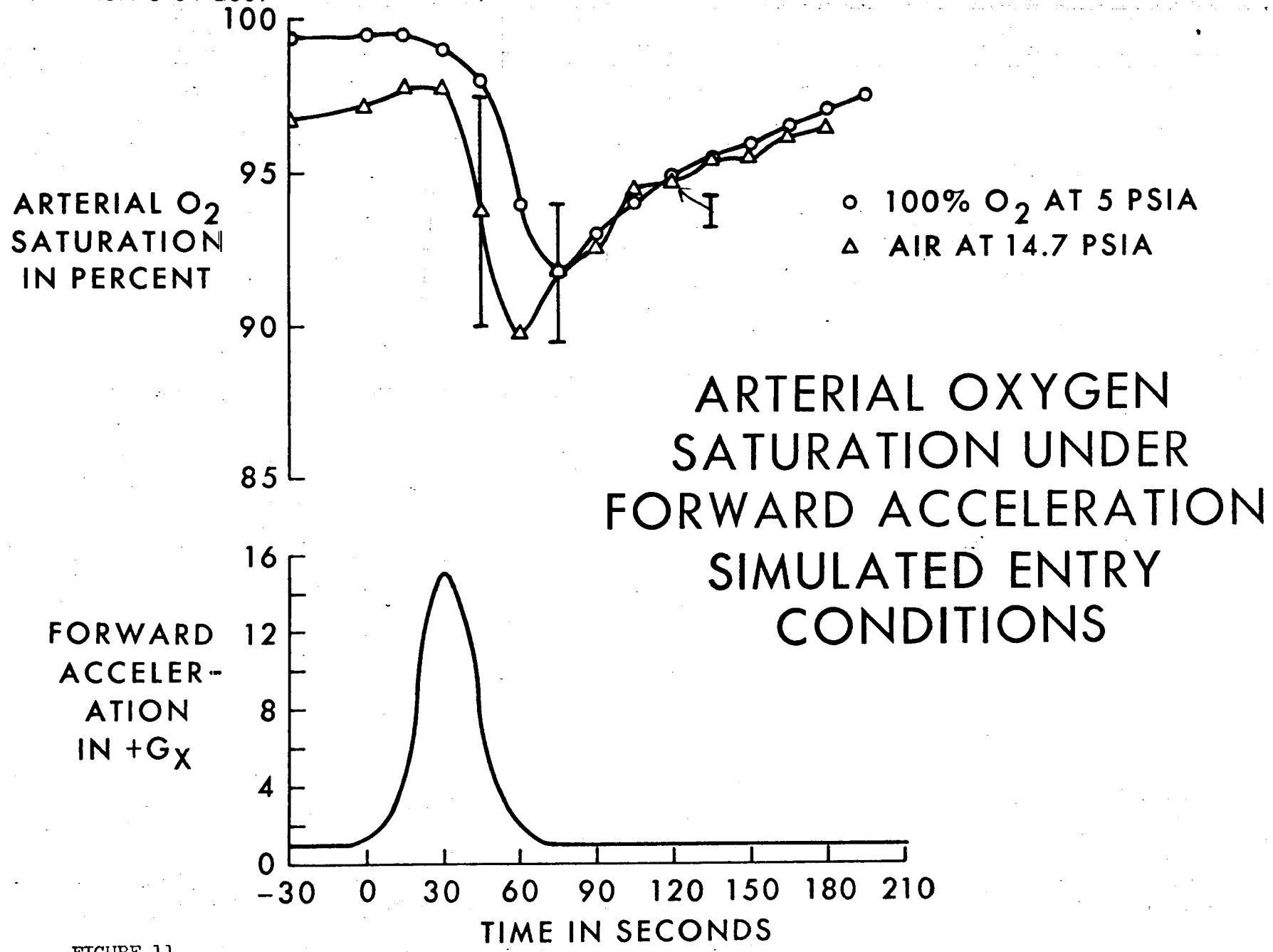


FIGURE 10

NASA-S-64-2609



ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION SIMULATED ENTRY CONDITIONS

FIGURE 11

NASA-S-64-2610

ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION SIMULATED ENTRY CONDITIONS

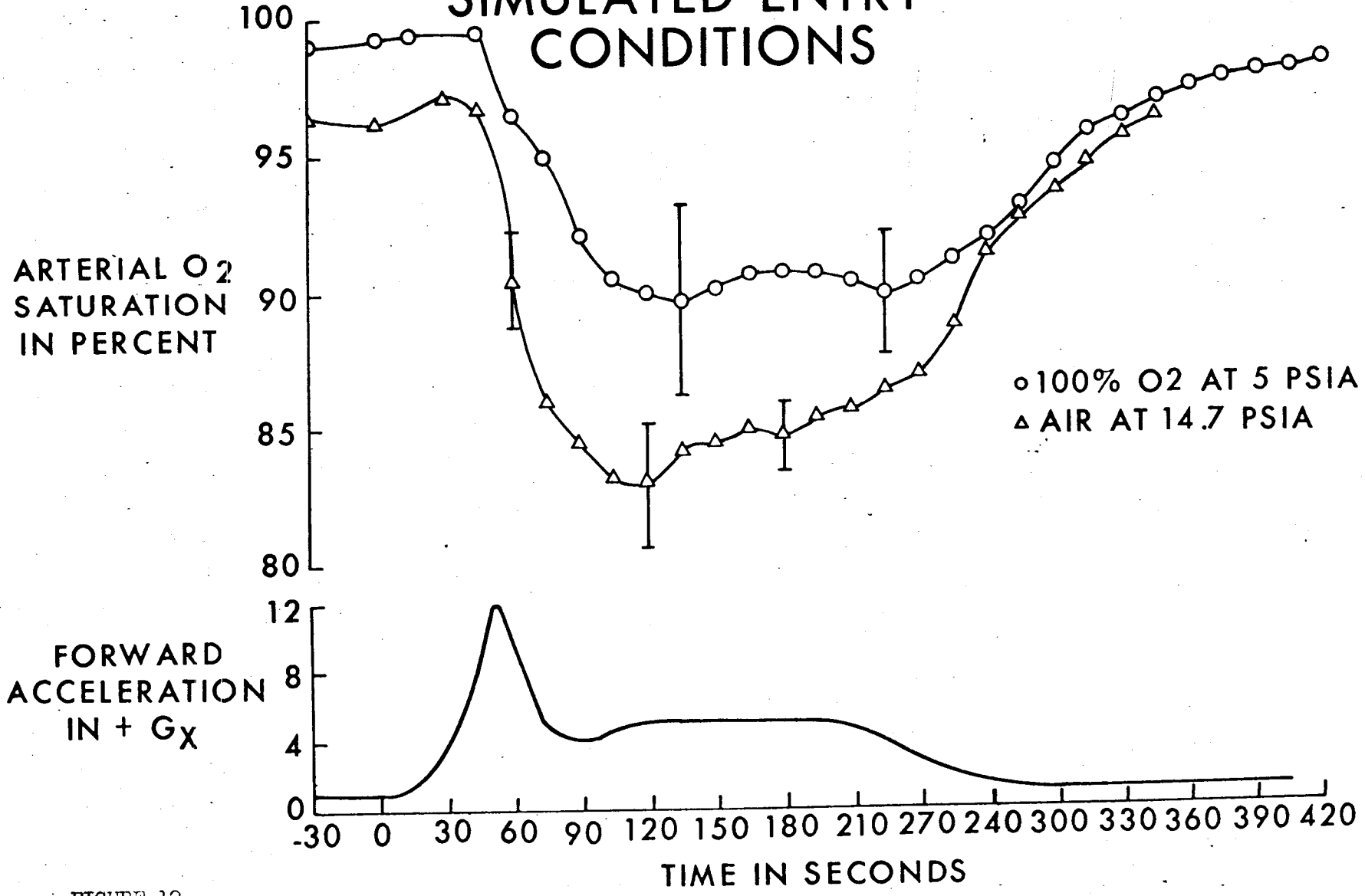


FIGURE 12

NASA-S-64-2611

ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION AIR AT 14.7 PSIA

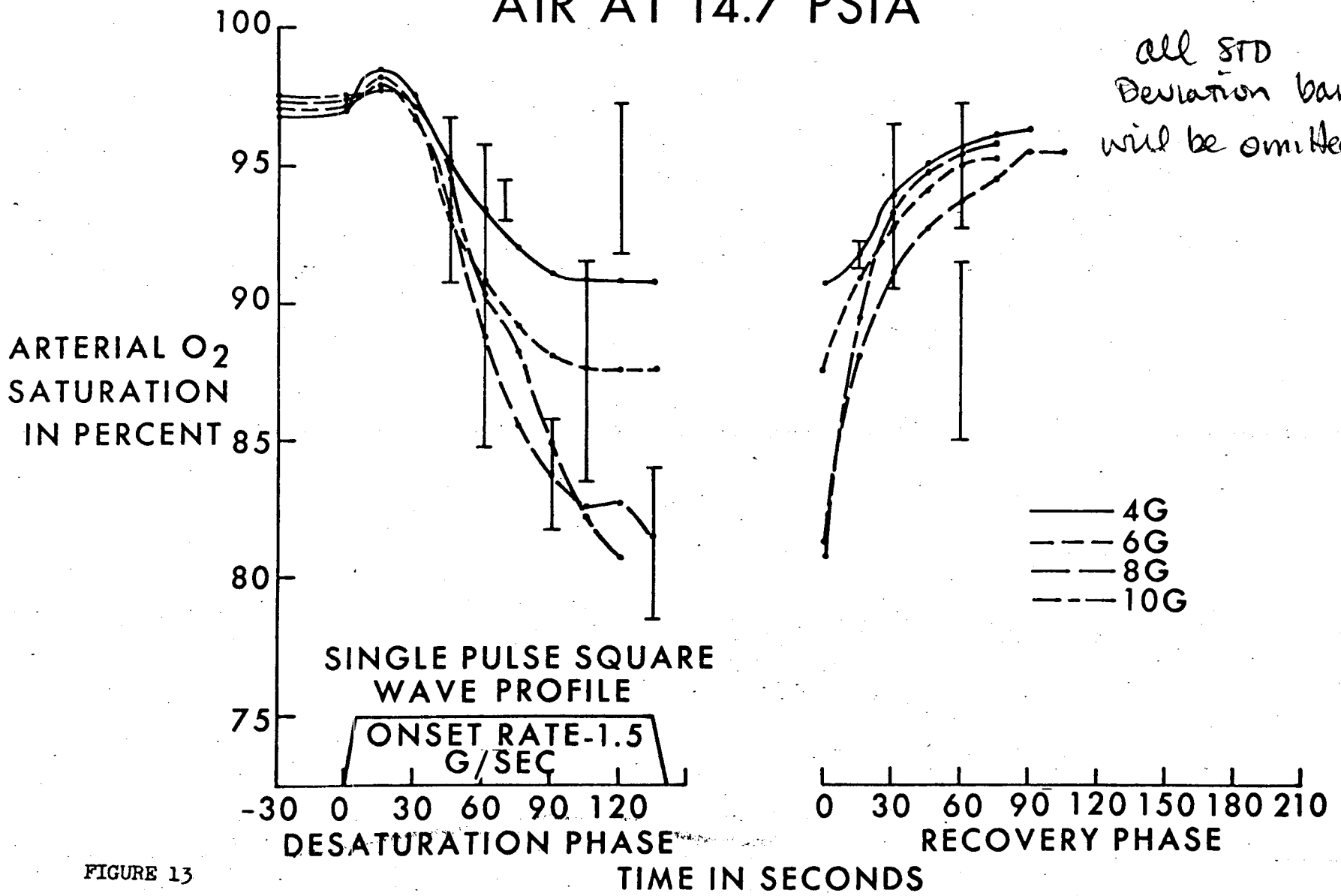


FIGURE 13

NASA-S-64-2612

ARTERIAL OXYGEN SATURATION UNDER FORWARD ACCELERATION PURE OXYGEN AT 5 PSIA

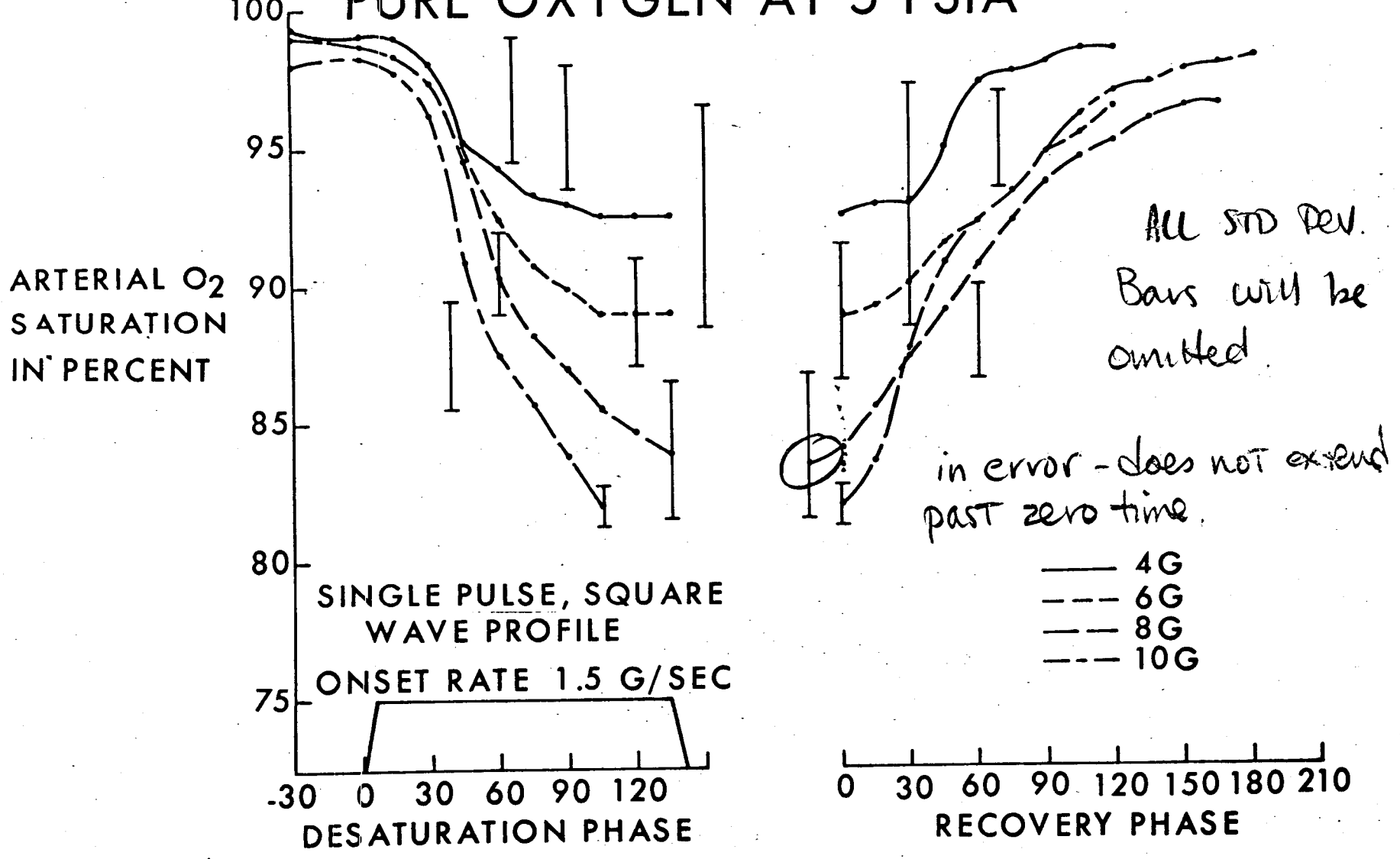


FIGURE 14